UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/835,298	04/13/2001	Jeffrey R. Dahlen	071959-5301	4762

30542 7590 06/04/2007

FOLEY & LARDNER LLP
P.O. BOX 80278
SAN DIEGO, CA 92138-0278

EXAMINER	
LAM, ANN Y	
ART UNIT	**PAPER NUMBER**
1641	

MAIL DATE	DELIVERY MODE
06/04/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

UNITED STATES PATENT AND TRADEMARK OFFICE

Commissioner for Patents
United States Patent and Trademark Office
P.O. Box 1450
Alexandria, VA 22313-1450
www.uspto.gov

BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Application Number: 09/835,298
Filing Date: April 13, 2001
Appellant(s): DAHLEN ET AL.

Barry Wilson
For Appellant

EXAMINER'S ANSWER

This is in response to the appeal brief filed March 6, 2007 appealing from the Office action mailed January 29, 2007.

(1) Real Party in Interest

A statement identifying by name the real party in interest is contained in the brief.

(2) Related Appeals and Interferences

The examiner is not aware of any related appeals, interferences, or judicial proceedings which will directly affect or be directly affected by or have a bearing on the Board's decision in the pending appeal.

(3) Status of Claims

The statement of the status of claims contained in the brief is correct.

(4) Status of Amendments After Final

The appellant's statement of the status of amendments after final rejection contained in the brief is correct.

(5) Summary of Claimed Subject Matter

The summary of claimed subject matter contained in the brief is correct.

(6) Grounds of Rejection to be Reviewed on Appeal

The appellant's statement of the grounds of rejection to be reviewed on appeal is correct.

(7) Claims Appendix

The copy of the appealed claims contained in the Appendix to the brief is correct.

(8) Evidence Relied Upon

5,290,678 Jackowski 3-1994

Antman et al., N. Engl. J. Med. 335:1342-49 (1996)

Richards et al., Heart 81:114-20 (1999)

(9) Grounds of Rejection

The following ground(s) of rejection are applicable to the appealed claims:

35 USC § 112, 6th Paragraph

The Office notes that the language "means for determining cardiac mortality rate" in claim 23, line 9, and claim 25, line 10, respectively, and "means for determining binding" in claim 23, line 8, and claim 25, line 8, respectively, are being treated under *35 USC § 112, 6th Paragraph.*

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 23-28, 32-34 and 38 are rejected under 35 U.S.C. 103(a) as being unpatentable over **Jackowski**, [5,290,678], in view of **Antman et al.**, ["Cardiac-specific Troponin I Levels to Predict the Risk of Mortality in Patients with Acute Coronary Syndromes", The New England Journal of Medicine, (1996), pp. 1342-1349, Vol. 335, No. 18], and further in view of **Richards et al.**, ["Neuroendocrine prediction of left ventricular function and heart failure after acute myocardial infarction", Heart, (1999); 81: 114-120].

Jackowski teaches the invention substantially as claimed. Jackowski teaches a multimarker approach comprising the use of antibodies for detecting the presence of at least three markers of cardiac damage in a patient's serum and that the combined

responses of reagents indicates the diagnostic condition of the patient (col. 5, lines 17-21, and lines 43-51).

Jackowski teaches that troponin may be one of the markers that may be detected for this purpose (col. 8, lines 28-29). However, Jackowski does not teach detecting the combination of troponin and BNP, nor for the purpose of detecting cardiac mortality.

Antman et al. however teach that cardiac troponin I can be measured by immunoassay using antibodies that recognize cardiac troponin I (see page 1343, left column, last paragraph.) Moreover, Antman et al. teach that cardiac troponin I in blood is an independent risk factor that identifies patients presenting with unstable angina or non-Q-wave myocardial infarction who are at increased risk of death (see page 1347, left col., last paragraph.) Thus, it would have been obvious to one of ordinary skill in the art at the time the invention was made to utilize the multimarker assay taught by Jackowski using cardiac troponin I to predict increased risk of death because Antman et al. teach that cardiac troponin I in blood is an independent risk factor that identifies patients presenting with unstable angina or non-Q-wave myocardial infarction who are at increased risk of death. One of ordinary skill in the art would recognize the medical benefits of detecting increased risk of death.

Moreover, Richards et al. teach that plasma BNP measured within 1 to 4 days of acute myocardial infarction is a powerful independent predictor of death over the subsequent 14 months (see page 114, left column, last paragraph under the heading "*Conclusions*"). Richards et al. collected blood samples from patients (see page 114, right col., last paragraph) and tested for cardiac peptides using an immunoassay (i.e., a

binding assay using antibodies), (see page 115, left col., 1st paragraph). The patients in the study had acute myocardial infarction (see table 3 on page 117). Moreover, Richards et al. teach that adding BNP in a multivariate analyses added additional information in predicting the composite end point of death (see page 118, right column, last paragraph). Richards et al. concluded that stratification of patients into low and high risk groups can be greatly facilitated by plasma BNP measurements and that these could be included in the routine clinical work up of patients following myocardial infarction (see page 119, right column, last paragraph, under the heading "CONCLUSION"). While Richards et al. do not specifically state that the same type of analysis, i.e., radioimmunoassay, as used in the experiment may be performed for clinical analyses, it is understood to be the same type, i.e., immunoassay (which uses antibodies). (Alternatively, it would have been obvious to one of ordinary skill in the art that the same type of assay, i.e., immunoassay, used by Richards et al. in the experiment may be used for clinical analyses because Richards et al. teach that BNP can be detected using immunoassays.)

It would have been obvious to one of ordinary skill in the art at the time the invention was made to provide BNP as taught by Richards et al. as a marker in the multimarker assay taught by Jackowski modified by Antman et al. for the purpose of predicting cardiac mortality rate in patients with acute myocardial infarction because Richards et al. teach that BNP is a powerful predictor of death in patients with acute myocardial infarction (see page 114, left column, last paragraph under the heading

"*Conclusions*") and that adding BNP as a marker to a multivariate analyses added additional information in predicting death (see page 118, right col., last paragraph).

To elaborate on the suggestions and motivations to combine the references, it is noted that Jackowski teaches a method of detecting more than one marker of cardiac damage (see col. 5, lines 18-20), the combined responses indicating the diagnostic condition of a patient (col. 5, lines 50-52). The basis for this multiple marker (or multimarker) approach is in part due to the fact that different markers of cardiac damage are released at different times (see for example, column 2, lines 58-63 and column 7, lines 47-50, disclosing that levels of CK-MB are not elevated until 6-8 hours after the onset of myocardial infarction and do not peak until after 12 hours and therefore its detection is not of use alone as a diagnostic test.) The basis for the multiple marker approach is also based on the usefulness of detecting multiple markers for determining different medical conditions related to cardiac damage (col. 5, lines 27-29, and col. 7, lines 47-67.) The invention of Jackowski thus utilizes different combinations of antibodies such that different cardiac markers are assessed, including markers that ensure detection of cardiac tissue damage at an early stage of patient chest pain (col. 8, lines 16-20), and markers that can detect myocardial infarction many hours after onset of chest pain where the patient is in the later stages of myocardial infarction (col. 8, line 34-38) for the purpose of detecting myocardial infarction or unstable angina (col. 7, lines 52-66.) In short, Jackowski teaches the basis for a multiple marker approach in detecting markers of cardiac damage. While Jackowski discloses specific cardiac markers but does not disclose BNP, and teaches use of the markers for the purpose of

detecting myocardial infarction or unstable angina (col. 7, lines 52-66), rather than for predicting cardiac mortality rate, one of ordinary skill in the art would nevertheless recognize that Jackowski teaches the general concept of a multiple marker approach for determining different medical conditions related to cardiac damage particularly where the markers are released and peak at different times. Moreover, Antman et al. and Richards et al. teach, respectively, that cardiac troponin I and BNP are predictors of death in patients with myocardial infarction (see Antman et al. page 1347, bottom of left column; and Richards et al., page 114, bottom of left column). While Jackowski does not teach a method of predicting increased risk of death, one of ordinary skill in the art would recognize the medical benefits of detecting increased risk of death taught by Antman et al. and Richards et al. Moreover, one of ordinary skill in the art would recognize the benefit of measuring troponin I as taught by Antman et al. and BNP as taught by Richards et al. in a multiple marker approach generally taught by Jackowski because Jackowski suggests that different cardiac markers are detectable and peak at different times after cardiac injury and Antman et al. and Richards et al. disclose different time frames for measuring troponin I and BNP for predicting death, with Antman et al. emphasizing that troponin I permits early identification of patients at increased risk of death, even where measured 6 hours or less after the onset of chest pain, and especially above 6 hours to 24 hours, and Richards et al. disclosing that BNP is powerful predictor of death 1-4 days after the onset of acute myocardial infarction. Furthermore, one of ordinary skill in the art would be suggested to measure troponin I as an early predictor of death, as taught by Antman et al., and BNP as a powerful

predictor of death, particularly because Richards et al. teach that measuring the level of BNP could reasonably be included in the routine clinical work up of patients following myocardial infarction, thus suggesting that measuring BNP levels should be coupled with other clinical procedures performed on patients with myocardial infarction. Thus, the disclosures of Antman et al. and Richards et al. provide motivations and suggestions for detecting troponin I and BNP in a multiple marker approach taught by Jackowski, either as additional markers to those disclosed by Jackowski, i.e., for the additional determination of a medical condition, specifically predicting likelihood of death, or as a separate multiple marker assay method for the purpose of predicting likelihood of death. One of ordinary skill in the art would recognize from the disclosure by Antman et al. and Richards et al. that detecting both troponin I and BNP provide the advantage of a better prediction of likelihood of death as these cardiac markers are disclosed as being detectable at different times, in light of the disclosure by Jackowski that a multiple marker approach provides more information and thus a better determination of a medical condition of a patient.

Thus, with respect to independent claims 23, 25, 27 and 33, Richards et al. teach the steps of contacting a sample with a second antibody (i.e., antibodies used in the radioimmunoassay on page 115, left col., 1st paragraph) that specifically binds to a second marker (BNP), (see page 118, right column, 1st full paragraph);

providing means for determining binding between each of said respective markers and each of said respective antibodies (i.e. the radioimmunoassay, page 115, left col., first paragraph),

whereby said binding provides a means for determining cardiac mortality rate (page 118, right col., last paragraph). (As to claims 27 and 33, the prognosis is considered to be cardiac mortality rate, or death.)

As to the following claims, the references teach the limitations as follows.

As to claims 24, 26, 28,and 34, said body fluid is blood (Antman et al., page 1343, left col., 3rd full paragraph; and Richards et al., page 114, right col., last paragraph).

Regarding the preamble in claims 23 and 27, the above method of predicting cardiac mortality rate is performed on a patient that *has* an acute coronary syndrome (see Antman et al., page 1347, left col., last paragraph; and see Richards et al. page 114, left column, last paragraph).

With respect to 32 and 38, the prognosis is considered to be mortality rate or subsequent death (see Antman et al., page 1347, left col., last paragraph; and see Richards et al. page 114, left column, last paragraph.)

Regarding the preamble in claims 25 and 33, while the references teach that the markers may be performed on patients *with* acute coronary syndromes, such as acute myocardial infarction (see above with respect to claims 23 and 27), the references however do not specifically state that the patients were actually *diagnosed* with acute coronary syndromes. However, the references suggest that a method of predicting mortality rate using the markers should be performed on those patients who have been diagnosed with acute coronary syndromes because they suggest the benefits of performing such a method on high risk groups (which would include those that actually

have been diagnosed with acute coronary syndrome). For example, Antman et al. teach that the disclosed method of predicting mortality permits the early identification of patients at increased risk of death (page 1348, right column, last paragraph). Moreover, Richards et al. suggest that stratification of patients into low and high risk groups can be greatly facilitated by plasma BNP measurements and that these could be included in the routine clinical work up of patients following myocardial infarction (see page 119, right column, last paragraph).

(10) Response to Argument

Appellants argue that Examiner's grounds for rejection are flawed and that even assuming a prima facie case of obviousness, Appellants' evidence demonstrating superior unexpected results of the present invention, industry acclaim, copying and adoption by others rebuts any such prima facie case.

First, Appellants argue that the primary reference, the Jackowski patent, is related to *diagnosis* of acute coronary syndromes, in contrast to Appellants' method claims, which are directed to a *prognosis*. Appellants emphasize that the two are different in that a diagnosis is the ability to distinguish the presence of a disease from its absence, and a prognosis looks only at those individuals having the disease and asks which are predisposed to suffer from an event of interest at some later point in time. Appellants argue that Examiner does not explain how the Jackowski method of using multiple markers for *diagnosing* unstable angina or myocardial infarction is relevant to the present claims, which relates to a *prognosis*.

Examiner acknowledges the differences between the Jackowski patent and the instant claims as recognized by Appellants. However, Jackowski teaches benefits of a multiple marker approach in a diagnosis which the skilled artisan would recognize as also being advantageous in a prognosis, particularly in light of the disclosures by Antman et al. and Richards et al. As explained in the last final Office action, Jackowski teaches that one advantage of using multiple markers is that it improves the testing for presence of markers that appear at different stages in the development of a biological condition, e.g., myocardial infarction. For example, Jackowski, in column 2, lines 58-63 and column 7, lines 47-50, discloses that levels of CK-MB are not elevated until 6-8 hours after the onset of myocardial infarction and do not peak until after 12 hours and therefore its detection is not of use alone as a diagnostic test. The basis for the multiple marker approach is also based on the usefulness of detecting multiple markers for determining different medical conditions related to cardiac damage, such as distinguishing between unstable angina and myocardial infarction (see Jackowski, col. 5, lines 27-29, and col. 7, lines 47-67.) The invention of Jackowski thus utilizes different combinations of antibodies such that different cardiac markers are assessed, including markers that ensure detection of cardiac tissue damage at an early stage of patient chest pain (col. 8, lines 16-20), and markers that can detect myocardial infarction many hours after onset of chest pain where the patient is in the later stages of myocardial infarction (col. 8, line 34-38) for the purpose of detecting myocardial infarction or unstable angina (col. 7, lines 52-66.)

In short, Jackowski teaches the basis for a multiple marker approach in detecting markers of cardiac damage. While Jackowski discloses specific cardiac markers but does not disclose BNP, and teaches use of the markers for the purpose of detecting myocardial infarction or unstable angina (col. 7, lines 52-66), rather than for predicting cardiac mortality rate, one of ordinary skill in the art would nevertheless recognize that Jackowski teaches the general concept of a multiple marker approach for determining different medical conditions related to cardiac damage particularly where the markers are released and peak at different times. The disclosures of Antman et al. and Richards et al. respectively show that troponin I and BNP are markers for prediction of death in patients with myocardial infarction (see *Antman et al.* page 1347, bottom of left column; and see *Richards et al.,* page 114, bottom of left column), and the disclosures in these references of *when* these markers can be detected for such predictions indicate that these markers appear and remain elevated at different time periods, as explained further below. Thus, the skilled artisan would recognize that the advantages taught by Jackowski in using a multiple marker approach in diagnosing for example myocardial infarction would also be advantageous in a prognosis of death in patients with myocardial infarction when the prognosis is based on testing for markers that are released and peak at different times. The disclosures of Antman et al. and Richards et al. suggest that a multiple marker approach generally taught by Jackowski for improving testing would also be beneficial for testing for troponin I and BNP to predict likelihood of death since troponin I and BNP markers are disclosed as being predictors of death that can be detected at *different* time periods. That is, the advantage of increasing the

chance of detecting at a particular point in time at least one marker of a biological condition of interest, such as a myocardial infarction as taught by Jackowski is also relevant to a method of predicting death since the markers for predicting death are disclosed in the prior art as being detected at different time periods.

More specifically, Antman et al. teach that troponin I is an independent risk factor that identifies patients presenting with unstable angina or non-Q-wave myocardial infarction who are at increased risk of death (page 1345 lower right column; and page 1347, bottom of left column), and that measurement of troponin I taken from as early as 0 to 6 hours after onset of chest pain show an increase in risk of mortality where the cardiac troponin I level is at least 0.4 ng/milliliter and that the prognostic value of cardiac troponin I was greatest during the time period greater than 6 to 24 hours after chest pain as compared to 0 to 6 hours and 0 to 24 hours (see figure 2 and also page 1348, second full paragraph.) It is also noted that Antman et al. teach that cardiac troponin I permits the *early* identification of patients at increased risk of death (page 1348, right column.)

Moreover, Richards et al. teach that plasma BNP measured 1 to 4 days of acute myocardial infarction is a powerful independent predictor of death over the subsequent 14 months (see page 114, left column, under *"Conclusion"*.). It is emphasized that Richards et al. teach that the level of BNP was predictive of death where BNP measurement was 1 to 4 days (see page 114, left column, under *"Design"* and also table 3). While Richards et al. indicate that the level of BNP added additional information beyond clinical features, noradrenaline concentrations, and LVEF in

predicting the composite end point of death (see page 118, right column, last paragraph), rather than in addition to the level of, for example, Troponin I, one of ordinary skill in the art, based on the teachings of Jackowski as well as Antman et al. and Richards et al., would recognize the benefit of a multiple marker approach to predicting death using Troponin I as taught by Antman et al. and BNP as taught by Richards et al., as Antman et al. and Richards et al. disclose different time frames for measuring troponin I and BNP for predicting death.

Thus, one of ordinary skill in the art would recognize the benefit of detecting the level of both troponin I and BNP as predictors of death because these markers are disclosed to be predictors of death at different times, troponin I, as early as 0 to 6 hours, and especially 6 to 24 hours after onset of chest pain, as taught by Antman et al., and BNP from 1 to 4 days after acute myocardial infarction, as taught by Richards et al. The skilled artisan would recognize that a multiple marker approach as taught by Jackowski for diagnosing myocardial infarction would also improve a testing for the prediction of death since the reason for testing for more than one marker is the same in both cases, that is, to increase the chance of detecting at least one marker of the biological condition of interest. Moreover, it is emphasized that Richards et al. also suggest measuring the level of BNP in addition to other routine clinical work up of a patient following myocardial infarction (page 119, right column, last paragraph). While it is not clear that "routine clinical work" includes measuring other markers of cardiac damage, Richards et al. nevertheless suggest measuring BNP levels as an additional test because it is a powerful predictor of death in patients with acute myocardial infarction.

In summary, Jackowski teach that a multiple marker approach provides more information and thus a better determination of a medical condition of a patient especially where markers are released and peak at different times, and thus one of ordinary skill in the art would recognize from the disclosure by Antman et al. and Richards et al. that detecting *both* troponin I and BNP provides the advantage of a better prognosis as these cardiac markers are disclosed as being detectable at different time periods for the prediction of likelihood of death.

In support of superior, unexpected results, Appellants point out that the superior properties of combining BNP and cardiac troponin for prognosis in acute coronary syndromes described in the present specification were later confirmed in the scientific literature, for example the *New England Journal of Medicine (NEJM),* which disclosed, among other things, that: the levels of traditional serum markers of myocyte necrosis, such as creatine kinase MB fraction and troponin I, is only partially successful in risk stratification; an elevated troponin level confers an increased short-term risk of death; and measurements of three markers significantly increased physicians' ability to detect acute coronary syndromes, as compared with the use of each marker alone. The *NEJM* issue also disclosed that a single measurement of B-type natriuretic peptide (BNP) obtained a median of 40 hours after the onset of ischemic symptoms predicted the risk of death in patients who had myocardial infarction, and that the relation between the long-term risk of death and the B-type natriuretic peptide level was independent of electrocardiographic changes, troponin levels, renal function and congestive heart failure.

Examiner however finds that the superior results obtained by detecting a combination of BNP and troponin levels, as compared to detecting levels of either markers alone, are already suggested by the prior art and thus are *not unexpected.* The teachings of all three references suggest that different markers provide additional information, with Jackowski teaching that where different markers are elevated and peak at different times, detection of only one marker may be insufficient, and Antman et al. teaching that elevated levels of troponin I correlates to increased risk of death even 0 to 6 hours after onset of chest pain, and more strongly correlates at 6 to 24 hours, and Richards et al. teaching a different time frame in measuring BNP, that is, 24 to 96 hours after onset of symptoms, for predicting death. Thus, *it is expected* that there is an increased likelihood of accurately predicting death since an assay for multiple markers for the prognosis increases the likelihood of detecting at least one of the markers at any point in time. Moreover, the disclosure by Antman et al. that troponin I is a marker for predicting death when detected between 0 to 24 hours after onset of chest pain, and the disclosure of Richards et al. that BNP is a marker for predicting death when detected 24 to 96 hours after onset of symptoms show that troponin I confers a relatively short-term risk of death and BNP confers a relatively long-term risk of death, and thus such findings are not unexpected.

Appellants also point out that other researchers, such as Omland et al., published articles concerning the biosynthetically related polypeptide NT-proBNP, demonstrating that NT-proBNP and cardiac troponin measurements also provide independent prognostic information in acute coronary syndromes, and that Sabatine et

al. reports that when used together in a combined strategy, BNP and cardiac troponin provide a more effective tool for identifying patients at increased risk for clinically important cardiac events related to heart failure and acute coronary syndrome. Thus, Appellants assert that Appellants' invention was met with acclaim and was quickly copied and adopted within the art, which thus rebut any prima facie case of obviousness. As to the Omland et al. reference, this disclosure does not rebut the obviousness rejection because it is not relevant to the rejection which is based on teachings of BNP, rather than proBNP or NT-proBNP, where the claims list BNP as one of the markers as an alternative to proBNP and NT-proBNP. As to the Sabatine et al. reference, it is not mentioned by Appellants to be specifically related to *prognosis of death*.

Appellants further assert that it was unexpected that BNP measurements and cardiac troponin measurements would provide independent prognostic information *across the entire spectrum of ACS conditions*, and not only in acute myocardial infarction. Appellants argue that the importance of Appellants' contribution in this regard has been widely recognized, acknowledged, and adopted in the art, and that the unexpected properties of the claimed invention, and the secondary considerations represented by the widespread approval and adoption of the claimed invention in the art, rebut any prima facie case of obviousness. Appellants assert that the art led one to believe that any information provided by BNP would be restricted to the subset of acute myocardial infarction. That is, Appellants maintain that it is unexpected that the claimed invention can provide prognostic information across the entire spectrum of acute

coronary syndromes (ACS) because the scientific literature at the time indicated that BNP measurements would not be applicable outside of the context of acute myocardial infarction. However while Appellants merely assert that BNP provides prognostic information *across the entire spectrum* of acute coronary syndromes, Appellants do not provide any evidence in support of this assertion.

Appellants also assert that the practical advantage of combining the measurements of BNP and cardiac troponin has been widely recognized, acknowledged, and adopted in the art, as demonstrated by report by Silver et al. (2004) prepared by an expert panel gathered by selecting clinicians and scientists with expertise with the natriuretic peptide system. To show the practical advantage of combined measurements of BNP and cardiac troponin, Appellants cited the report's statement that when used together in a combined strategy, BNP and cardiac troponin provide a more effective tool for identifying patients at increased risk for clinically important cardiac events related to HF [presumably heart failure] and acute coronary syndrome, and that multimarker panels that include BNP, troponin, and C-reactive protein are now available and each of these markers provides unique and independent information with regard to patient outcomes. Appellants' citation of the report however does not indicate that cardiac events includes death, and thus does not provide direct evidence related to Appellants' claims, which are directed to predicting likelihood of death. Moreover, as already discussed above, the advantages of detecting a combination of BNP and cardiac troponin is *not unexpected*. As mentioned above, the advantage of improving a test by increasing the chance of detecting at least one marker

of a biological condition of interest, such as a myocardial infarction as taught by Jackowski, is also advantageous in a method of predicting death since the markers for predicting death are disclosed in the art as being detected at different time periods.

Lastly, it is noted that Appellants' independent claims 23 and 25 do not recite a step of correlating a measured amount of BNP and troponin to a prognosis of death, and thus a prior art disclosure of detecting BNP and troponin meets these claims.

(11) Related Proceeding(s) Appendix

No decision rendered by a court or the Board is identified by the examiner in the Related Appeals and Interferences section of this examiner's answer.

For the above reasons, it is believed that the rejections should be sustained.

Respectfully submitted,

Ann Lam

LONG V. LE
SUPERVISORY PATENT EXAMINER
TECHNOLOGY CENTER 1600

Conferees:

Long Le

Larry Helms

LARRY R. HELMS, PH.D.
SUPERVISORY PATENT EXAMINER